

[First Hit](#) [Fwd Refs](#)[Previous Doc](#)[Next Doc](#)[Go to Doc#](#)**End of Result Set**

Generate Collection

Print

L2: Entry 1 of 2

File: USPT

Dec 30, 2003

US-PAT-NO: 6669941

DOCUMENT-IDENTIFIER: US 6669941 B1

TITLE: Soluble lymphotoxin-.beta. receptor as a therapeutic agent for treating TH-1 cell-associated autoimmune disease

DATE-ISSUED: December 30, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Browning; Jeffrey L.	Brookline	MA		
Benjamin; Christopher D.	Beverly	MA		
Hochman; Paula S.	Brookline	MA		

US-CL-CURRENT: 424/192.1; 514/2, 514/8, 514/825, 514/866, 514/885, 514/903

CLAIMS:

What is claimed is:

1. A method for treating or reducing the advancement, severity or effects of a Th1 cell-associated autoimmune disease in an animal comprising the step of administering a pharmaceutical composition which comprises a therapeutically effective amount of a soluble lymphotoxin-.beta. receptor (LT-.beta.-R) fused to one or more heterologous protein domains and a pharmaceutically acceptable carrier.
2. The method according to claim 1, wherein the animal is a mammal.
3. The method according to claim 2, wherein the mammal is a human.
4. The method according to claim 1, wherein the soluble LT-.beta.-R comprises a ligand binding domain that can selectively bind to a surface LT ligand.
5. The method according to claim 4, wherein the heterologous protein domain comprises a human immunoglobulin Fc domain.
6. The method according to any one of claims 1-4 wherein the Th1 cell-associated autoimmune disease is rheumatoid arthritis.
7. The method according to claim 5 wherein the Th1 cell-associated autoimmune disease is rheumatoid arthritis.
8. The method according to any one of claims 1-4 wherein the Th1 cell-associated autoimmune disease is multiple sclerosis.

9. The method according to claim 5 wherein the Th1 cell-associated autoimmune disease is multiple sclerosis.

10. The method according to any one of claims 1-4 wherein the Th1 cell-associated autoimmune disease is diabetes.

11. The method according to claim 5 wherein the Th1 cell-associated autoimmune disease is diabetes.

12. A pharmaceutical composition comprising a therapeutically effective amount of a soluble lymphotoxin-.beta. receptor (LT-.beta.-R) fused to one or more heterologous protein domains and a pharmaceutically acceptable carrier.

13. The composition according to claim 12, wherein the soluble LT-.beta.-R comprises a LT-.beta.-R ligand binding domain that can selectively bind to a surface LT ligand.

14. The composition according to claim 13, wherein the heterologous protein domain comprises a human immunoglobulin Fc domain.

[Previous Doc](#)

[Next Doc](#)

[Go to Doc#](#)

[First Hit](#) [Fwd Refs](#)[Previous Doc](#)[Next Doc](#)[Go to Doc#](#)**End of Result Set**

Generate Collection

Print

L1: Entry 1 of 2

File: USPT

Jun 11, 2002

US-PAT-NO: 6403087

DOCUMENT-IDENTIFIER: US 6403087 B1

TITLE: Soluble lymphotoxin-.beta. receptors as therapeutic agents for the treatment of immunological disease

DATE-ISSUED: June 11, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Browning; Jeffrey L.	Brookline	MA		
Benjamin; Christopher D.	Beverly	MA		
Hochman; Paula S.	Newton	MA		

US-CL-CURRENT: [424/134.1](#); [424/133.1](#), [514/2](#), [514/8](#), [530/387.1](#), [530/387.3](#)

CLAIMS:

What is claimed is:

1. A method for inhibiting a Th1 cell-mediated immune response in an animal comprising the step of administering a pharmaceutical composition which comprises an effective amount of a soluble lymphotoxin-.beta.-receptor (LT-.beta.-R) fused to one or more heterologous protein domains and a pharmaceutically effective carrier.
2. The method according to claim 1, wherein the animal is a mammal.
3. The method according to claim 2, wherein the mammal is a human.
4. The method according to claim 1, wherein the soluble LT-.beta.-R comprises a ligand binding domain that can selectively bind to a surface LT ligand.
5. The method according to claim 1, wherein the heterologous protein domain comprises a human immunoglobulin Fc domain.
6. The method according to claim 1, wherein the Th1 cell-mediated immune response contributes to a delayed type hypersensitivity reaction.
7. The method according to claim 6, wherein the delayed type hypersensitivity reaction is contact hypersensitivity.
8. The method according to claim 6, wherein the delayed type hypersensitivity reaction is tuberculin-type hypersensitivity.

9. The method according to claim 6, wherein the delayed type hypersensitivity reaction is a granulomatous reaction.

10. The method according to claim 1, wherein the Th1 cell-mediated immune response contributes to cellular rejection of tissue in the animal after the animal receives a tissue graft.

11. The method according to claim 1, wherein the Th1 cell-mediated immune response contributes to organ rejection in the animal after the animal receives an organ transplant.

12. The method according to claim 1, wherein the Th1 cell-mediated immune response contributes to an autoimmune disorder in the animal.

13. The method according to claim 12, wherein the autoimmune disorder is selected from the group consisting of multiple sclerosis, insulin-dependent diabetes, sympathetic ophthalmia, uveitis and psoriasis.

14. The method according to claim 1, wherein the Th1 cell-mediated immune response is inhibited without inhibiting a Th2 cell-dependent immune response.

[Previous Doc](#)

[Next Doc](#)

[Go to Doc#](#)